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Comparative Analysis of Sodium Excretion and CKD Progression: Insights from CRIC and KNOW-CKD Cohorts

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Objectives : Urinary sodium levels, reflecting salt intake, have been linked to the development of cardiovascular disease and chronic kidney disease (CKD). Excessive sodium consumption may exacerbate CKD, through indirect mechanisms, such as blood pressure (BP) elevation or via direct nephrotoxic effects. However, the extent to which these associations vary by ethnicity remains unclear.
Methods : This study included 5,655 participants with CKD G2–4 from two prospective CKD cohorts: the CRIC study in the United States and the KNOW-CKD study in South Korea. The primary exposure was 24-hour urinary sodium excretion. The primary outcome included CKD progression, defined as ≥50% decline of baseline estimated glomerular filtration rate or initiation of kidney failure with replacement therapy. Cause-specific models were employed to evaluate to the relationship between 24-hour urinary sodium excretion and the risk of CKD. Causal mediation analysis was conducted to quantify the extent to which systolic blood pressure mediated this relationship.
Results : Over a median follow-up of 5.43 years (39,085 person-years), the primary outcome occurred in 1,896 patients, with an incident rate of 48.5 per person years. Participants in the highest quartile exhibited a greater risk of CKD progression compared to those in the lowest quartile. The adjusted HRs (95% CIs) for CKD progression were 1.28 (1.06–1.55) in the CRIC cohort and 2.08 (1.33–3.24) in the KNOW-CKD cohort. Mediation analysis revealed that systolic BP accounted for 24% of the observed effect on CKD progression in the CRIC cohort, compared to only 1% in the KNOW-CKD cohort.
Conclusions : Higher urinary sodium excretion was associated with increased CKD progression in both cohorts. However, mechanistic pathways differed by ethnicity: systolic BP mediated a greater effect in CRIC, whereas KNOW-CKD showed a more direct, BP-independent effect. These findings suggest ethnic and physiological differences in sodium-related kidney injury and underscore the need for personalized dietary interventions in CKD management.

table 1_ UNa.png

Table 1. Hazard ratios for the CKD progression according to quartiles of 24hr urine sodium excretion

		Quartiles of 24hr Urine Sodium Excretion			
		Q1	Q2	Q3	Q4
CKD progression		HR (95% CI)	HR (95% CI)	HR (95% CI)	HR (95% CI)
Overall cohort					
	Model 1	Reference	0.89 (0.78, 1.02)	1.06 (0.94, 1.21)	1.04 (0.91, 1.17)
	Model 2	Reference	0.93 (0.81, 1.08)	1.15 (1.01, 1.32)	1.23 (1.06, 1.42)
	Model 3	Reference	1.07 (0.92, 1.25)	1.22 (1.05, 1.43)	1.45 (1.22, 1.72)
	Model 4	Reference	1.06 (0.91, 1.24)	1.18 (1.01, 1.39)	1.37 (1.15, 1.63)
CRIC					
	Model 1	Reference	0.98 (0.83, 1.16)	1.14 (0.97, 1.34)	1.09 (0.93, 1.28)
	Model 2	Reference	0.99 (0.83, 1.17)	1.17 (0.99, 1.38)	1.17 (0.98, 1.40)
	Model 3	Reference	1.06 (0.89, 1.27)	1.19 (1.00, 1.41)	1.37 (1.13, 1.65)
	Model 4	Reference	1.06 (0.89, 1.26)	1.13 (0.95, 1.35)	1.28 (1.06, 1.55)
KNOW-CKD					
	Model 1	Reference	0.75 (0.60, 0.95)	0.94 (0.76, 1.16)	1.00 (0.80, 1.24)
	Model 2	Reference	0.87 (0.67, 1.12)	1.23 (0.96, 1.57)	1.53 (1.17, 1.99)
	Model 3	Reference	0.87 (0.61, 1.26)	1.41 (0.98, 2.03)	1.96 (1.33, 2.90)
	Model 4	Reference	0.98 (0.67, 1.44)	1.55 (1.06, 2.28)	2.08 (1.33, 3.24)

Model 1: unadjusted
Model 2: + age, sex, BMI, race, 24hr urine creatinine, systolic blood pressure, alcohol status, smoking status, education, medical history (diabetes, cardiovascular disease)
Model 3: + natural log 24hr urine potassium, eGFR, serum albumin, serum hemoglobin, HDL cholesterol, serum phosphorus
Model 4: + use of medications (RAS blocker, diuretics, statin)

table 1_ UNa.png

Table 2. The effect of urine sodium excretion on CKD progression via systolic blood pressure

Outcome	Mediator	HR of 24hr Urine sodium excretion on CKD progression	%Mediation[a]
Overall cohort			
CKD progression	Systolic blood pressure		
	Total effect	1.46 (1.19, 1.81)	
	Direct effect	1.40 (1.13, 1.73)	
	Indirect effect	1.05 (1.02, 1.09)	16%
CRIC cohort			
CKD progression	Systolic blood pressure		
	Total effect	1.40 (1.10, 1.73)	
	Direct effect	1.32 (1.04, 1.62)	
	Indirect effect	1.07 (1.03, 1.13)	24%
KNOW-CKD cohort			
CKD progression	Systolic blood pressure		
	Total effect	2.09 (1.09, 3.35)	
	Direct effect	2.08 (1.08, 3.36)	
	Indirect effect	1.01 (0.94, 1.06)	1%

Models are adjusted for age, sex, BMI, 24hr urine creatinine, primary renal disease, race, education, smoking status, alcohol status, history of cardiovascular disease, natural log 24hr urine potassium, eGFR, serum albumin, serum phosphorus, HDL cholesterol, hemoglobin, use of medications (RAS blocker, diuretics, statin), including systolic blood pressure as a mediator.
[a]Calculated as Direct Effect x (Indirect Effect - 1)/(Total Effect - 1).